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Effects of environmental tobacco smoke on the respiratory health of children

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This review synthesizes current knowledge of the effects of prenatal and postnatal exposure to environmental tobacco smoke on the respiratory health of children. A Medline database search was conducted for 1966 through October 2000. Limited evidence was found that exposure in pregnancy influences fetal growth, increases the risk of preterm delivery, and predicts the development of asthma and reduced lung function later in life. Both occupational and home environments contribute to the exposure of pregnant women and thus indirectly to adverse effects on children. There is strong and consistent evidence that exposure in childhood causes chronic respiratory symptoms (eg, cough, phlegm, and wheezing) and induces asthma. Limited evidence supports the role of childhood exposure in the poor overall control of established disease. Postnatal exposure is likely to have a small adverse impact on lung function growth. Prenatal and postnatal exposures have an important impact on children's respiratory health. These effects are preventable if pregnant women and children are protected from exposure to environmental tobacco smoke.

Key terms asthma, children, chronic obstructive pulmonary disease, lung function, pregnancy, respiratory infections, respiratory symptoms, review, tobacco smoke pollution.

In this paper, we review the evidence of the effects of environmental tobacco smoke on the respiratory health of children, consider questions for future research, and discuss public health issues and possibilities for prevention. In another paper in this journal, we present the corresponding evidence for adults (1). We conducted a Medline database search from 1966 through October 2000 with the Mesh-terms "Tobacco smoke pollution and exp. Respiratory tract diseases". Additional material was collected by systemically reading the reference lists of articles and by relying on personal knowledge of research under way. The literature on the respiratory effects of environmental tobacco smoke on children is extensive. This review summarizes the evidence from a series of meta-analyses and reviews (2–12) and complements it with relevant new findings. We also consider directions for further research, public health impact, and possibilities for prevention.

The influence of parental smoking on a child's respiratory health begins during the fetal period and continues through infancy and childhood. Maternal smoking in pregnancy can be considered a type of prenatal fetal exposure to environmental tobacco smoke. A pregnant woman's exposure to environmental tobacco smoke constitutes another type of prenatal exposure. After birth both parents and other household members contribute to the child's exposure. The constituents of environmental tobacco smoke from parents and household members are naturally similar, but the mother's closeness with the child often results in higher exposure per cigarette smoked. Prenatal and postnatal exposures may be related, and therefore it is difficult to separate their

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effects on a child. The effects related to maternal smoking have often, but not consistently, been found to be stronger than those related to paternal smoking (9–12). This finding can be explained by the closer presence of a mother with her child or by the influence of a mother's smoking in pregnancy. The type, level, and timing of exposure are important when the adverse effects of environmental tobacco smoke on respiratory health are assessed (13). We present separately the evidence on the effects of maternal smoking in pregnancy and exposure to environmental tobacco smoke in pregnancy and in childhood.

Effects of maternal smoking in pregnancy

There is both direct and indirect evidence that smoking in pregnancy adversely influences fetal development of the respiratory system. Foundas and his colleagues (14) showed that, in early pregnancy, smoking mothers' urinary cotinine levels correspond to those of any active smoker. This finding indicates that similar metabolic processes take place also during pregnancy. Due to the free diffusion of cotinine through body fluids and the placenta, fetal exposure levels may be similar to those of active smokers. The adverse effects of smoking during pregnancy on fetal growth are well-established (15). Low birthweight is a predictor of subsequent respiratory disease in childhood, such as asthma (16). These two findings are consistent with the important role of a mother's smoking with respect to the respiratory health of her child. In a Danish study of 4795 male conscripts, the adjusted odds ratio (OR) for asthma related to birthweights below 2501 grams was 1.5 [95% confidence interval (95%CI) 0.7–3.1] in comparison with that of conscripts with birthweights of 3001–3500 grams (16).

Findings of a relation between maternal smoking in pregnancy and lung function impairment in newborns provide evidence of actual effects on the respiratory system (17–22). Two studies, one conducted in Australia (17) and the other in Norway (18), reported that newborns of smoking mothers have a lower ratio of time-to-maximal-tidal-expiratory-flow to total time of expiration (tPTEF/tE) when compared with newborns of nonsmoking mothers. Although the estimated effects on lung function may not have clinical significance in infancy, they indicate a detectable pathophysiological process as a result of maternal smoking. In the Norwegian study of 802 newborns, the compliance of airways at birth was also decreased (18). The effect on fetal development takes place well before birth, since similar findings have been reported for preterm infants (19). Airway hyperresponsiveness has been reported to be increased soon after birth among infants of mothers who smoked in pregnancy (22).

Effects of environmental tobacco smoke on pregnant women

People exposed to environmental tobacco smoke mainly encounter the same compounds as in the mainstream smoke inhaled directly by the smoker, although the concentrations and time-patterns differ (11, 23). Therefore the fetus is also exposed to tobacco smoke products when the pregnant woman is exposed. Although individual studies have often been inconclusive, a recent meta-analysis by Windham and his colleagues (24) suggests that exposure to environmental tobacco smoke during pregnancy has a small adverse effect on birthweight and it increases the risk of term low birthweight. The evidence of an effect on preterm delivery is weaker (24), but the existence of such an effect is supported by the findings of two recent studies (25, 26). In a Californian study of 4454 singleton live births, high exposure to environmental tobacco smoke (≥7 hours/day for nonsmokers) was moderately associated with preterm birth with an adjusted odds ratio of 1.6 (95% CI 0.87–2.9) (25). A Finnish investigation included 389 nonsmoking women selected from a population-based study in southeast Finland on the basis of questionnaire information after delivery (response rate 94%) to represent exposure and nonexposure to passive smoking in pregnancy (26). The final exposure assessment was based on the nicotine concentration of maternal hair, which was sampled after the delivery. This concentration indicates exposure during the past two months (ie, in the third trimester). The exposure categories were defined a priori as high (nicotine concentration >4.00 μg/g, N=52) and medium (0.75–4.00 μg/g, N=186), with low (<0.75 μg/g, N=151) as the reference category. In a logistic regression analysis controlling for confounding, the risk of preterm delivery (<37 weeks) was higher in the high (adjusted OR 6.12, 95% CI 1.31–28.7) and medium (adjusted OR 1.30, 95% CI 0.30–5.58) exposure categories than in the reference category, and there was an increase of 1.22 (95% CI 1.07–1.39) in the adjusted odds ratio with a 1 μg/g nicotine increase in exposure. The corresponding adjusted odds ratio was 1.06 (95% CI 0.96–1.17) for low birthweight and 1.04 (95% CI 0.92–1.19) for small for gestational age. According to the self-reported exposure indices, work exposure was a stronger determinant of adverse pregnancy outcomes than home exposure was.

Surprisingly few studies have evaluated the effects of a mother's passive smoking during pregnancy on the lung function of the newborn or on children later in

childhood. The aforementioned Norwegian study found no effects of passive smoking in pregnancy on the newborn's tPTEF:tE or airway compliance (18). The Six Cities Study reported a reduction in forced expiratory volume in 0.75 seconds ($FEV_{0.75}$) at the age of 8–12 years in relation to active smoking of a mother in pregnancy, but not to the passive smoking of a mother (27).

Effects of environmental tobacco smoke in childhood

Respiratory symptoms and asthma

The first reports of an effect of parental smoking on children's respiratory symptoms were published in the early 1970s (28–30). Since then, many studies have examined the effects of exposure to environmental tobacco smoke in childhood (12). Strachan & Cook (2) conducted a meta-analysis of seven studies of wheezing illness in early childhood. Maternal smoking increased the risk of wheezing with a summary odds ratio of 1.98 (95% CI 1.71 – 2.30), but the study-specific estimates showed substantial heterogeneity (P=0.049). In infancy and early childhood, wheezing is often closely related to acute respiratory illnesses, including bronchitis, pneumonia, and bronchiolitis. Therefore, some studies have combined wheezing and lower respiratory tract infections to form the construct "lower respiratory illness". Although wheezing in early childhood does not always lead to the development of chronic asthma, it is a strong predictor of an increased risk of childhood asthma. In a cohort study from Tuscon, 60% of the children experiencing wheezing had recovered by school age (31).

A large number of studies has provided evidence of an effect of environmental tobacco smoke on respiratory symptoms in school-aged children. Strachan & Cook (3) summarized the results of population-based cross-sectional studies. There were 41 on wheezing (32–69), 34 on chronic cough (28, 30, 32, 33, 36–40, 42–49, 51, 58–59, 61–62, 64, 67, 70–77), 7 on chronic phlegm (32, 37–38, 43, 46, 48, 65), and 6 on breathlessness (40, 46, 49, 57, 62,70) (table 1). The risk of wheezing was associated with either parent smoking, the summary odds ratio being 1.24 (95% CI 1.10–1.34). The corresponding odds ratio was 1.40 (95% CI 1.27–1.53) for cough, 1.35 (95% CI 1.13–1.62) for phlegm, and 1.31 (95% CI 1.08–1.59) for breathlessness. The measures of effect were lower for one parent smoking and higher for both parents smoking and therefore indicated an exposure-dependent effect. The effect of maternal smoking was higher than that of paternal smoking. This finding could be explained either by higher levels of exposure from maternal smoking or by the effects of smoking during pregnancy.

A working group of the California Environmental Protection Agency (11) conducted a meta-analysis of 37 studies assessing the effect of postnatal exposure to environmental tobacco smoke from household sources on the development of asthma, wheezy bronchitis, or constant wheezing among children younger than 18 years of age. These investigations included cross-sectional, longitudinal, and case-referent studies (37, 39, 41, 43, 45, 48, 49, 51–52, 60, 62–63, 71, 74, 75, 78–99). The odds ratio of the random-effects summary was 1.44 (95% CI 1.27–1.64) for clinically diagnosed asthma and 1.47 (95% CI 1.34–1.61) for wheezy bronchitis or chronic wheezing. The measure of effect was similar for younger and older children (cut point 10 years of age). Strachan & Cook (3) summarized studies according to the study design. In a meta-analysis of 21 population-based cross-sectional studies published by April 1997, the risk of asthma was related to the smoking of either parent with an odds ratio of 1.21 (95% CI 1.10–1.34). The effect estimate was 1.50 (95% CI 1.29–1.73) when both parents smoked and 1.04 (95% CI 0.78–1.38) when only one parent smoked. The odds ratio was 1.36 for mother's smoking and 1.07 for father's smoking. The development of asthma-like symptoms and asthma may lead to parents avoiding smoking in the presence of their children. The presence of these symptoms may also influence the reporting of exposure to environmental tobacco smoke. Thus the cross-sectional study design is sensitive to selection and information bias, both being likely to reduce the effect estimate. Strachan & Cook (6)

Table 1. Estimates, from a meta-analysis (3), of the relationship between parental smoking and respiratory symptoms among school-age children. (OR = odds ratio, calculated with the random effects model, 95% CI = 95% confidence interval, NC = not calculated due to the small number of studies)

Symptom	Either parent smoking		Mother only smoking		Father only smoking		Both parents smoking	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Wheezing	1.24	1.17–1.31	1.28	1.19–1.38	1.14	1.06–1.23	1.47	1.14–1.90
Cough	1.40	1.27–1.53	1.40	1.20–1.64	1.21	1.09–1.34	1.67	1.48–1.89
Phlegm	1.35	1.13–1.62	NC	·	NC	·	1.46	1.04–2.05
Breathlessness	1.31	1.08–1.59	NC	·	NC	·	NC	·

identified eight longitudinal studies (85, 92, 100–103) that assessed the incidence of asthma or wheezing rather than the prevalence of asthma as the outcome. The incidence of asthma or wheezing was related to maternal smoking, but the effect was stronger for the first 5–7 years of age (4 studies, summary odds ratio 1.31, 95% CI 1.22–1.41) than for school age (4 studies, summary OR 1.13, 95% CI 1.04–1.22). The two longitudinal studies of asthma or asthma-like symptoms published after this meta-analysis provide additional evidence of the effect of early-life exposure to environmental tobacco smoke (104–105). In a 2-year cohort study of 3754 children born in Oslo, the risk of bronchial obstruction was greater for children exposed to environmental tobacco smoke than for unexposed children, with an adjusted odds ratio of 1.6 (95% CI 1.3–2.1) (104). The effect was seen in relation to both maternal and paternal smoking alone. In a cohort study of 499 children of asthmatic or allergic parents from Boston, the risk of repeated wheezing episodes during the first 12 months was related to maternal smoking during pregnancy with a relative risk of 1.83 (95% CI 1.12–3.00) (105). Adding paternal smoking did not add to the predictive power of maternal smoking.

Several studies carried out in Australia, Europe, and the United States provide strong evidence that exposure to environmental tobacco smoke causes increased reactivity of the airways in children (7). A meta-analysis of studies of bronchial hyperresponsiveness suggests that the excess risk due to exposure to environmental tobacco smoke in different populations is relatively weak (29%), but however not explained by chance (7, 9). This finding refers to populations not restricted to those with some predisposing characteristic, such as a family history of asthma.

There is accumulating evidence that exposure to environmental tobacco smoke influences the prognosis of asthma in childhood. Strachan & Cook (6) identified eight studies on the effect of parental smoking on the development of wheezing from infancy to school age (31, 82, 102, 106–110). Five studies (31, 82, 106, 108–109), focusing on the effects of exposure to environmental tobacco smoke on the persistence of early wheezing in childhood, reported a summary odds ratio of 1.35 (95% CI 0.87–2.08). Parental smoking worsened the recovery from parainfluenza bronchiolitis by increasing the number of subsequent wheezing episodes (107). However, in two large British cohort studies, maternal smoking was associated with a significantly reduced risk of having asthma or bronchitis at ages from 11 and 23 years among persons who had asthma by the age of 7 years (102, 110). In these studies other sources of environmental tobacco smoke, which may have greater importance at older ages, were not taken into account. For example, parents of children with severe asthma are more likely to quit smoking.

A study of the relation between exposure to environmental tobacco smoke and the severity of asthma is complicated for several reasons. The indicators of asthma severity have several dimensions, including symptoms and signs of asthma on one hand and the need for medication on the other. The outcome may influence the avoidance of exposure, and the use of medication may be related both to the exposure and to the outcome. Three recent reviews (8, 11–12) assessed the effects of environmental tobacco smoke on the severity or exacerbation of asthma in children; no formal meta-analysis was conducted due to the heterogeneity of the outcome assessment. These reviews concluded that exposure to environmental tobacco smoke increases disease severity, as assessed by the frequency and intensity of asthma attacks, the number of emergency room visits during a year, the use of asthma medication, and the occurrence of asthma attacks requiring intubation. Six studies used hyperresponsiveness of the airways in the challenge tests or diurnal variability in peak expiratory flow (PEF) as the measure of asthma severity (111–117). These studies indicate an increased diurnal PEF variability in relation to exposure to environmental tobacco smoke.

Lung function

In 1977, Schilling and his colleagues published the first epidemiologic study, of 816 children, to assess the relation between parental smoking and children's lung function (33). Since then, over 40 cross-sectional studies (27, 37, 39–40, 43, 46–47, 49, 53, 61–62, 71, 92, 115, 117–143) and 6 longitudinal studies (37, 49, 134, 144–146) have been published. Table 2 summarizes the effect estimates from a meta-analysis of the cross-sectional studies (8) and from the two largest longitudinal studies (144, 146), which contribute most of the information.

Four (37, 49, 145–146) of six longitudinal studies reported an adverse effect of passive smoking on lung function. By far the largest study, of 8706 schoolchildren in six cities of the United States, included up to 12 annual lung function measurements and collected detailed information on passive and active smoking (146). Current maternal smoking was related to slower growth rates of FEV_1 (–3.8 ml/year, 95% CI –6.4 – –1.1), FVC (–2.8 ml/year, 95% CI –5.45 – 0.0), and FEF_{25-75} [–14.3 ml/(s·year), 95% CI –29.0 – 0.3].

Two systematic quantitative reviews indicate that children of smoking parents have a small deficit in lung function when measured by forced expiratory volume in one second (FEV_1) and midexpiratory flow (MEF) (8–9, 11). In the most recent meta-analysis, based on 21

cross-sectional studies, Cook and his colleagues (8) reported a 1.4% (95% CI 1.0–1.9) lower FEV_1 for exposed children than for those unexposed. A total of 10 studies (27, 46, 53, 84, 119–120, 122, 124, 132, 136) allowed a comparison of the effects of maternal and paternal smoking, and the effect of maternal smoking was predominantly stronger. Only one study, from China, has reported a substantial effect of paternal smoking (122). Usually the effect of both parents' smoking was stronger than mother's smoking alone. This finding could be explained by the additive effect of the exposures or by a higher degree of smoking among women whose spouses smoked. In the 19 applicable studies, the overall effect on forced vital capacity (FVC) was –0.4% (95% CI –0.8 – 0.0), and that on MEF was –5.0% (95% CI –6.6 – –3.3).

The finding of maternal smoking as a stronger determinant of lung function than the smoking of the father or other household members could be explained by exposure in utero due to the mother's smoking during pregnancy or by closer contact of the child with the mother than with other household members. Some of the studies tried to assess the effect of in utero exposure by collecting information on maternal smoking habits during pregnancy, but it was difficult to separate the effects of prenatal and postnatal exposures because they are often highly correlated. The large study conducted in 22 cities in the United States was able to identify a sufficient number of mothers who had smoked either during pregnancy only or before or after pregnancy only. Children 8 to 12 years of age whose mothers had smoked during pregnancy only had significantly lower levels of $FEV_{0.75}$ (–1.1%, 95% CI –2.1 – –0.2) and FEF_{25-75} (–4.4%, 95% CI –6.7 – –2.1), but not of FEV_1 (–0.7, 95% CI –1.7 – 0.2) when compared with children of never smoking mothers (27). The effect of prenatal exposure combined with maternal smoking during the past year was slightly stronger for all the lung function parameters: FEV_1 –1.6% (95% CI –2.2 – –1.0), $FEV_{0.75}$ –2.0% (95% CI –2.6 – –1.4), and FEF_{25-75} – 5.6% (95% CI –7.0 – –4.1). Maternal smoking during the past year, but not before, had no effect on lung function. These results suggest that prenatal and early-life exposures are more important than exposure during school age. Two (140, 146) out of three available cohort studies (134, 140, 146) provide evidence of the important role of mother's smoking during pregnancy or early childhood.

Some studies have addressed gender (27, 43, 62, 92, 117, 119, 122, 124, 140) and asthma as possible indicators of susceptibility to the effects of passive smoking. In seven of nine studies (27, 43, 62, 92, 117, 119, 122, 124, 140), the effect of environmental tobacco smoke on FEV_1 was stronger among boys than among girls. The summary estimate of the effect on FEV_1 was –2.1% (95% CI –2.8 – –1.5 %) for boys and –1.3% (95% CI –2.0 – –0.6 %) for girls (8). Two studies found greater effects on FEV_1 or midexpiratory flow rates among asthmatics (120, 132), while one reported greater effects among nonasthmatics (115).

Table 2. Estimated effect of parental smoking on ventilatory lung function among school-age children in longitudinal studies [from two individual studies (144, 146)] and in cross-sectional studies [from a meta-analysis (8)]. (95% CI=95% confidence interval, FEF_{25-75}=mean forced expiratory flow during the middle half of forced vital capacity, FEV_1=forced expiratory volume in 1 second, FVC=forced vital capacity, MEF=midexpiratory flow)

Lung function parameter	Number of studies [a]	Estimated effect [b]	95% CI [b]
Longitudinal studies			
FVC	1 (146)	-2.8 ml/year [c]	-5.5 – 0.0
FEV_1	1 (146)	-3.8 ml/year [c]	-6.4 – -1.1
FEF_{25-75}	1 (146)	-14.3 ml/(s·year) [c]	-29.0 – 0.3
$FEV_{1.0}$	1 (144)	-27.8 ml/year [d]	-50.1 – -5.5
Cross-sectional studies			
FVC	19	-0.4% [e]	-0.8 – 0.0
FEV_1	21	-1.4%	-1.9 – -1.0
MEF	19	-5.0%	-6.6 – -3.3

[a] Reference number in parentheses.
[b] The estimated effect and 95% CI have been calculated with the random effects model.
[c] Estimate given for an average exposure of one pack per day.
[d] Mother smoker versus nonsmoker or ex-smoker.
[e] The difference in lung function level between the exposed and unexposed, expressed as a percentage of the level of the unexposed group.

Respiratory infections

A large number of studies of the effects of environmental tobacco smoke in infancy and early childhood has focused on the construct "lower respiratory tract illness", which combines respiratory infections such as acute bronchitis, bronchiolitis, respiratory syncytial virus infections, and pneumonia, and sometimes also symptoms of the lower respiratory tract, such as wheezing and cough. The evidence consistently shows that exposure to environmental tobacco smoke increases the risk of lower respiratory illness early in life. A meta-analysis of 24 studies in a community setting yielded a summary odds ratio of 1.57 (95% CI 1.42–1.74) for smoking by either parent and 1.72 (95% CI 1.55–1.91) for maternal smoking (2).

There are fewer studies on specific infections of either the upper or lower respiratory tract. Four population-based cohort studies assessed the effect of environmental tobacco smoke on acute bronchitis and pneumonia in early childhood (100, 147–149). In a birth cohort of 2074 infants from the United Kingdom the adjusted odds ratio of bronchitis or pneumonia was 1.96 (95% CI 1.38–2.80) for either parent smoking and 2.79 (95% CI 1.87–4.15) for both parents smoking (147). For a cohort

of 1114 children up to 2 years age from New Zealand, the corresponding risk estimates were 1.56 (95% CI 1.15–2.12) and 1.83 (95% CI 1.22–2.74), respectively (100). In an 18-month follow-up of a cohort of infants in Shanghai, the People's Republic of China, the risk of pneumonia or bronchitis increased with the number of cigarettes smoked in the household (148). The odd ratio was 1.3 for 1–9 cigarettes smoked per day, 1.7 for 10–19 cigarettes smoked per day, and 2.0 for 20–39 cigarettes smoked per day (P for trend = 0.0002).

The results from a Norwegian birth cohort of 3754 children indicate that the effects of environmental tobacco smoke on susceptibility to infections can be protected, at least to some extent, by breastfeeding the child for a lengthy period. The risk of lower respiratory tract infections during the first year of life, including acute bronchitis, pneumonia, pseudo croup, and respiratory syncytial virus, was compared between children in the following four categories: environmental tobacco smoke and short breastfeeding; no environmental tobacco smoke and short breastfeeding (0–6 months), environmental tobacco smoke and long breastfeeding (>6 months); and no environmental tobacco smoke and long breastfeeding (reference category). The adjusted odds ratio for environmental tobacco smoke and short breastfeeding was 1.9 (95% CI 1.3–2.7), but for environmental tobacco smoke and long breastfeeding it was only 1.0 (95% CI 0.6–1.5).

There is strong evidence that exposure to environmental tobacco smoke increases the risk of middle-ear disease in children. Uhari and his colleagues (150) conducted a meta-analysis of the effects of exposure to environmental tobacco smoke on acute otitis media using studies from 1966 to 1994. The summary risk ratio was 1.66 (95% CI 1.33–2.06). Strachan & Cook (4) presented a meta-analysis based on nine studies of recurrent otitis media (151–159), four prevalence surveys of middle-ear effusion (160–163), and nine studies of middle-ear effusion based on surgical referrals (164–172). The odds ratio of the fixed-effects summary for the effect of either parent smoking on recurrent otitis media was 1.41 (95% CI 1.19–1.66), but the study-specific estimates were heterogeneous (P=0.036). The corresponding estimate for prevalent middle-ear effusion was 1.38 (95% CI 1.23–1.55), and for the clinic referral studies it was 1.21 (95% CI 0.95–1.53).

Summary of the evidence

Smoking in pregnancy

There is strong evidence that maternal smoking during pregnancy adversely influences fetal growth, and limited evidence indicates that it increases the risk of preterm delivery. Findings indicating that low birthweight predict the development of asthma later in life and results in reduced lung function are consistent with the role of maternal smoking during pregnancy in connection with adverse respiratory health effects. Limited, more-direct evidence indicates that smoking in pregnancy harms the fetal development of the respiratory system, increases the risk of asthma, and reduces the growth of lung function later in the life of the child.

Exposure to environmental tobacco smoke during pregnancy

There is strong evidence that exposure to environmental tobacco smoke during pregnancy increases the risk of low birthweight and preterm delivery. Exposure to environmental tobacco smoke during pregnancy may predict the development of asthma and reduced lung function later in life, but more studies on these issues are needed.

Exposure to environmental tobacco smoke during childhood

The evidence of the effects of exposure to environmental tobacco smoke during childhood is summarized in table 3. The judgment of causality is based on the number of studies, their validity, the evidence of dose-response relations, and biological plausibility. There is strong, consistent evidence that exposure to environmental tobacco smoke in childhood causes chronic respiratory symptoms, such as cough, phlegm, and wheezing. Strong evidence also supports a causal role of environmental tobacco smoke in childhood asthma, especially in the induction of asthma, but also in the poor overall control of established disease. The evidence is convincing that parental, especially maternal, smoking is related to small lung function deficits in neonates and schoolchildren. Some studies suggest that maternal smoking during pregnancy has a stronger effect than postnatal exposure, but this question has not yet been definitely resolved. Longitudinal studies provide evidence that postnatal exposure is likely to have a small adverse impact on lung function growth. The long-term clinical significance of these small effects is not known, but they may predispose to additional lung insults at older ages. Exposure to environmental tobacco smoke is an established cause of sensitivity to lower respiratory infections, and also to acute and recurrent otitis media.

Discussion

Questions for further research

The majority of the evidence on the respiratory effects of exposure to environmental tobacco smoke is based on cross-sectional studies or prevalent case-referent studies. Although the causal evidence is strong for many conditions, the quantification of effect estimates may be weakened by problems related to cross-sectional design, such as selection and information bias. Thus longitudinal studies are needed. The development of objective measures of exposure to environmental tobacco smoke will also improve the validity of effect estimates. Hair nicotine concentration is a new and promising biomarker of exposure and has been applied in epidemiologic studies only recently (26). Proximal 2-cm lengths of hair provide a reasonable assessment for exposure over the past 2 months. Repeated collection of hair samples over time, combined with focused chemical analysis to satisfy efficiently the needs of the study, is a feasible approach for cohort studies.

Identification of the susceptible age periods and induction periods for different respiratory effects will improve our understanding of the adverse effects of environmental tobacco smoke. Recent findings point to the importance of fetal and early life exposure. Only few studies have been able to separate the effects of prenatal exposure from those of postnatal exposure (9).

Genetic susceptibility and the interaction of environmental tobacco smoke with other environmental exposures will constitute an extensive research area in the future. Two recent studies indicate that both heredity and other environmental exposures modify the effects of environmental tobacco smoke on asthma. In a Swedish case-referent study of 193 children with asthma and 318 referents aged 1–4 years (173), exposure to environmental tobacco smoke was associated with an excess risk for asthma (OR 1.7, 95% CI 1.1–2.3), and signs of home dampness tended to increase this risk (OR 1.3, CI 0.9–2.0). High exposure to cats or dogs resulted in an increased risk for those sensitized to cats or dogs (OR 2.7, 95% CI 1.0–7.3). A combination of exposure to environmental tobacco smoke, damp housing, and high exposure to cats or dogs was associated with an odds ratio of 8.0 (95% CI 1.9–34.1). In a Norwegian birth cohort study (174), parental atopy alone increased the risk of bronchial obstruction (OR 1.62, 95% CI 1.10–2.40) and asthma (OR 1.66, 95% CI 1.08–2.54). For children without parental atopy, there was little effect of exposure to environmental tobacco smoke on bronchial obstruction (OR 1.29, 95% CI 0.88–1.89) or asthma (OR 0.84, 95% CI 0.53–1.34). The presence of both parental atopy and exposure had a substantial effect both on bronchial obstruction (OR 2.88, 95% CI 1.91–4.32) and on asthma (OR 2.68, 95% 1.70–4.22). The results are consistent with the hypothesized modification of effects of environmental exposure by genetic constitution (so-called gene-environment interaction), and they suggest that some genetic markers could indicate susceptibility to environmental factors.

Table 3. Summary of postnatal exposure to environmental tobacco smoke and respiratory diseases and conditions among children. (95% CI = 95% confidence interval, EF = effect estimate, OR = odds ratio, FEV_1 = forced expiratory volume in 1 second)

Disease or condition	Age range[a]	OR[b,c] or EF[d,e]	95% CI[b]	Causality[f]
Chronic respiratory symptoms	School age			+++
Wheezing		OR 1.28	1.19–1.38	
Cough		OR 1.40	1.20–1.64	
Phlegm		OR 1.35	1.13–1.62	
Breathlessness		OR 1.31	1.08–1.59	
Asthma				
Induction				+++
Cross-sectional studies	School age	OR 1.36	1.20–1.55	
Case-referent studies	1–18 years	OR 1.59	1.27–1.99	
Longitudinal studies	< 3 years	OR 2.08	1.59–2.71	
	5–7 years	OR 1.31	1.22–1.41	
	1–17 years	OR 1.13	1.04–1.22	
Bronchial hyperresponsiveness	School age	OR 1.29	1.10–1.50	
Prognosis	Infancy-school age	OR 1.35	0.87–2.08	++
	School age-young adulthood	OR 0.71	0.57–0.89	0
Severity	0-17 years	Summary estimate not available		++
Lower respiratory illness	0-2 years	OR 1.72	1.55–1.91	+++
Acute otitis media	Infancy-school age	OR 1.66	1.33–2.06	+++
Recurrent otitis media	Infancy-school age	OR 1.41	1.19–1.66	+++
Lung function, FEV_1				
Cross-sectional studies	School age (8)	EF -1.4%[g]	-1.9 – -1.0	++
Longitudinal studies	School age			
Six Cities Study (146)		EF -3.8 ml/year[h]	-6.4 – -1.1	
East Boston Study (144)		EF -27.8 ml/year[i]	-50.1 – -5.5	

[a] Reference number in parentheses.
[b] OR and 95% CI from the meta-analysis (8).
[c] The OR for all diseases and conditions except FEV_1 as lung fuction parameter is given for maternal smoking versus neither parent smoking if such an estimate is available; otherwise it is given for either parent smoking versus neither parent smoking.
[d] Estimate of effect and 95% CI the from meta-analysis (8) and two individual studies (144, 146).
[e] The effect estimate is given for maternal smoking versus neither parent smoking if such an estimate was available; otherwise it is given for either parent or both parents smoking versus neither parent smoking.
[f] Causality as judged by the authors. Symbols: +++ = causal relation established, ++ = strong evidence of a causal relation, + = some evidence of a causal relation, 0 = no clear evidence of a causal relation.
[g] The difference in FEV_1 level between the exposed and unexposed, expressed as a percentage of the level of the unexposed group.
[h] Estimate given for an average exposure of one pack per day.
[i] Mother smoker versus nonsmoker or ex-smoker.

Longitudinal studies of bronchial hyperreactivity are needed to understand the development of asthmatic tendency, as well as the subsequent development of the condition. Several studies have shown that parental smoking is related to a poor overall control of asthma in children. The evidence would be strengthened by longitudinal studies that apply methods that objectively measure reactivity of the airways (eg, serial measurements of airway hyperresponsiveness in challenge tests or diurnal PEF variability). Well-planned intervention studies aiming at assessing the effects of preventing exposure to environmental tobacco smoke among asthmatic children would make an important contribution to our knowledge.

Public health impact and prevention

Recent reports from England (175) and Finland (176) indicate that smoking in pregnancy continues to be high in spite of the efforts made in health education. In England, the prevalence of smoking during pregnancy was 26% in 1997, and it did not change substantially between 1992 and 1997 (175). Although the prevalence of daily smoking among adult men in Finland decreased between 1985 and 1997, from 32% to 30%, that of smoking among women increased from 17% in 1985 to 20% in 1997 (177). This increase was reflected in the findings of smoking in pregnancy, which remained similar in 1987–1997 (15%). The prevalence of smoking was alarmingly high among young (37%), single (30%), and less-educated women (25%).

The results from England and Finland suggest that current practices to prevent smoking during pregnancy are not working or that there is a lack of sufficient prioritization for effective implementation.

One of the most important challenges for the future is to develop effective preventive measures that are suitable for different cultures. Since young children seem to be especially vulnerable to the harmful effects of environmental tobacco smoke, pregnant women and the parents of young children should be a special target group for preventive interventions. Intervention studies aiming at helping pregnant women stop smoking have provided encouraging results (178). A Finnish study provided evidence that children's exposure to environmental tobacco smoke depends on the education of the parents, the social situation of the family (single parenting being a risk factor), and knowledge of the child's health status (atopic disease being a protective factor) (179). These findings indicate that educating parents with respect to the health effects of environmental tobacco smoke and emphasizing the benefits of a smoke-free environment for the child, as well as supporting the social situation, can have an important impact on preventing the harmful consequences of exposure to environmental tobacco smoke.

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